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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	DEC 23	New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/ USPAT2
NEWS	4	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS	5	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS	6	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS	7	JAN 17	IPC 8 in the WPI family of databases including WPIFV
NEWS	8	JAN 30	Saved answer limit increased
NEWS	9	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS	10	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS	11	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS	12	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS	13	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS	14	FEB 28	TOXCENTER reloaded with enhancements
NEWS	15	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS	16	MAR 01	INSPEC reloaded and enhanced
NEWS	17	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS	18	MAR 08	X.25 communication option no longer available after June 2006
NEWS	19	MAR 22	EMBASE is now updated on a daily basis
NEWS	20	APR 03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS	21	APR 03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL
NEWS	22	APR 04	STN AnaVist $500 visualization usage credit offered
NEWS	23	APR 12	LINSPEC, learning database for INSPEC, reloaded and enhanced
NEWS	24	APR 12	Improved structure highlighting in FQHIT and QHIT display in MARPAT
NEWS	25	APR 12	Derwent World Patents Index to be reloaded and enhanced during second quarter; strategies may be affected

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
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=> s melusin
L1 75 MELUSIN

=> s L1 and trasngenic
L2 0 L1 AND TRASNGENIC

=> s L1 and transgenic
L3 4 L1 AND TRANSGENIC

=> d L3 all

L3 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2005:334635 BIOSIS
DN PREV200510121520
TI Cardiac overexpression of **melusin** protects from dilated cardiomyopathy due to long-standing pressure overload.
AU De Acetis, Marika; Notte, Antonella; Accornero, Federica; Selvetella, Giulio; Brancaccio, Mara; Vecchione, Carmine; Sbroggio, Mauro; Collino, Federica; Pacchioni, Beniamina; Lanfranchi, Gerolamo; Aretini, Alessandra; Ferretti, Roberta; Maffei, Angelo; Altruda, Fiorella; Silengo, Lorenzo; Tarone, Guido [Reprint Author]; Lembo, Giuseppe
CS Univ Turin, Dept Genet Biol and Biochem, Via Santena,5Bis, I-10126 Turin, Italy
guido.tarone@unito.it; lembo@neuromed.it
SO Circulation Research, (MAY 27 2005) Vol. 96, No. 10, pp. 1087-1094. CODEN: CIRUAL. ISSN: 0009-7330.
DT Article
LA English
ED Entered STN: 31 Aug 2005
Last Updated on STN: 31 Aug 2005
AB We have previously shown that genetic ablation of **melusin**, a muscle specific beta 1 integrin interacting protein, accelerates left ventricle (LV) dilation and heart failure in response to pressure

overload. Here we show that **melusin** expression was increased during compensated cardiac hypertrophy in mice subjected to 1 week pressure overload, but returned to basal levels in LV that have undergone dilation after 12 weeks of pressure overload. To better understand the role of **melusin** in cardiac remodeling, we overexpressed **melusin** in heart of **transgenic** mice. Echocardiography analysis indicated that **melusin** over-expression induced a mild cardiac hypertrophy in basal conditions (30% increase in interventricular septum thickness) with no obvious structural and functional alterations. After prolonged pressure overload (12 weeks), **melusin** overexpressing hearts underwent further hypertrophy retaining concentric LV remodeling and full contractile function, whereas wild-type LV showed pronounced chamber dilation with an impaired contractility. Analysis of signaling pathways indicated that **melusin** overexpression induced increased basal phosphorylation of GSK3 beta and ERK1/2. Moreover, AKT, GSK3 beta and ERK1/2 were hyper-phosphorylated on pressure overload in **melusin** overexpressing compared with wild-type mice. In addition, after 12 weeks of pressure overload LV of **melusin** overexpressing mice showed a very low level of cardiomyocyte apoptosis and stromal tissue deposition, as well as increased capillary density compared with wild-type. These results demonstrate that **melusin** overexpression allows prolonged concentric compensatory hypertrophy and protects against the transition toward cardiac dilation and failure in response to long-standing pressure overload.

CC Cytology - General 02502
Cytology - Animal 02506
Biochemistry studies - General 10060
Pathology - General 12502
Cardiovascular system - Physiology and biochemistry 14504
Cardiovascular system - Heart pathology 14506
Muscle - Physiology and biochemistry 17504

IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Cardiovascular System (Transport and Circulation)

IT Parts, Structures, & Systems of Organisms
cardiomyocyte: muscular system, circulatory system; muscle: muscular system; left ventricle: circulatory system; stromal tissue

IT Diseases
heart failure: heart disease
Heart Failure, Congestive (MeSH)

IT Diseases
dilated cardiomyopathy: heart disease, pathology
Cardiomyopathy, Congestive (MeSH)

IT Chemicals & Biochemicals
ERK1/2; AKT; GSK3-beta; **melusin**: expression

IT Methods & Equipment
echocardiography: laboratory techniques, diagnostic techniques, clinical techniques, imaging and microscopy techniques; genetic ablation: laboratory techniques, genetic techniques

IT Miscellaneous Descriptors
pressure overload; capillary density; cardiac remodeling; interventricular septum thickness

ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
mouse (common): **transgenic**
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

=> d L3 1-4 all

L3 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2005:334635 BIOSIS
DN PREV200510121520
TI Cardiac overexpression of **melusin** protects from dilated cardiomyopathy due to long-standing pressure overload.
AU De Acetis, Marika; Notte, Antonella; Accornero, Federica; Selvetella, Giulio; Brancaccio, Mara; Vecchione, Carmine; Sbroggio, Mauro; Collino, Federica; Pacchioni, Beniamina; Lanfranchi, Gerolamo; Aretini, Alessandra; Ferretti, Roberta; Maffei, Angelo; Altruda, Fiorella; Silengo, Lorenzo; Tarone, Guido [Reprint Author]; Lembo, Giuseppe
CS Univ Turin, Dept Genet Biol and Biochem, Via Santena,5Bis, I-10126 Turin, Italy
guido.tarone@unito.it; lembo@neuromed.it
SO Circulation Research, (MAY 27 2005) Vol. 96, No. 10, pp. 1087-1094.
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DT Article
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ED Entered STN: 31 Aug 2005
Last Updated on STN: 31 Aug 2005
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cardiomyocyte: muscular system, circulatory system; muscle: muscular system; left ventricle: circulatory system; stromal tissue
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heart failure: heart disease
Heart Failure, Congestive (MeSH)
IT Diseases

dilated cardiomyopathy: heart disease, pathology
Cardiomyopathy, Congestive (MeSH)
IT Chemicals & Biochemicals
ERK1/2; AKT; GSK3-beta; **melusin**: expression
IT Methods & Equipment
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Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
mouse (common): **transgenic**
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

L3 ANSWER 2 OF 4 MEDLINE on STN
AN 2005276325 MEDLINE
DN PubMed ID: 15860758
TI Cardiac overexpression of **melusin** protects from dilated cardiomyopathy due to long-standing pressure overload.
AU De Acetis Marika; Notte Antonella; Accornero Federica; Selvetella Giulio; Brancaccio Mara; Vecchione Carmine; Sbroggio Mauro; Collino Federica; Pacchioni Beniamina; Lanfranchi Gerolamo; Aretini Alessandra; Ferretti Roberta; Maffei Angelo; Altruda Fiorella; Silengo Lorenzo; Tarone Guido; Lembo Giuseppe
CS Department of Genetics, Biology, Turin University, Turin, Italy.
SO Circulation research, (2005 May 27) Vol. 96, No. 10, pp. 1087-94.
Electronic Publication: 2005-04-28.
Journal code: 0047103. E-ISSN: 1524-4571.
CM Erratum in: Circ Res. 2005 Jul 8;97(1):e5
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200510
ED Entered STN: 28 May 2005
Last Updated on STN: 12 Oct 2005
Entered Medline: 11 Oct 2005
AB We have previously shown that genetic ablation of **melusin**, a muscle specific beta 1 integrin interacting protein, accelerates left ventricle (LV) dilation and heart failure in response to pressure overload. Here we show that **melusin** expression was increased during compensated cardiac hypertrophy in mice subjected to 1 week pressure overload, but returned to basal levels in LV that have undergone dilation after 12 weeks of pressure overload. To better understand the role of **melusin** in cardiac remodeling, we overexpressed **melusin** in heart of **transgenic** mice. Echocardiography analysis indicated that **melusin** over-expression induced a mild cardiac hypertrophy in basal conditions (30% increase in interventricular septum thickness) with no obvious structural and functional alterations. After prolonged pressure overload (12 weeks), **melusin** overexpressing hearts underwent further hypertrophy retaining concentric LV remodeling and full contractile function, whereas wild-type LV showed pronounced chamber dilation with an impaired contractility. Analysis of signaling pathways indicated that **melusin** overexpression induced increased basal phosphorylation of GSK3beta and ERK1/2. Moreover, AKT, GSK3beta and ERK1/2 were hyper-phosphorylated on pressure overload in **melusin** overexpressing compared with wild-type mice. In addition,

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CT Animals
Apoptosis
Blood Pressure
Cardiomyopathy, Dilated: ET, etiology
*Cardiomyopathy, Dilated: PC, prevention & control
Cytoskeletal Proteins: GE, genetics
*Cytoskeletal Proteins: PH, physiology
Fibrosis
Glycogen Synthase Kinase 3: ME, metabolism
Humans
Hypertrophy, Left Ventricular: ET, etiology
Mice
Mice, Transgenic
Mitogen-Activated Protein Kinase 1: PH, physiology
Mitogen-Activated Protein Kinase 3: PH, physiology
Muscle Proteins: GE, genetics
*Muscle Proteins: PH, physiology
*Myocardium: ME, metabolism
Myocardium: PA, pathology
Myocytes, Cardiac: PA, pathology
Phosphorylation
Protein-Serine-Threonine Kinases: ME, metabolism
Proto-Oncogene Proteins: ME, metabolism
Proto-Oncogene Proteins c-akt
Rats
Rats, Sprague-Dawley
Research Support, Non-U.S. Gov't
Ventricular Remodeling

CN 0 (Cytoskeletal Proteins); 0 (Itgb1bp2 protein, mouse); 0 (Muscle Proteins); 0 (Proto-Oncogene Proteins); EC 2.7.1.37 (AKT1 protein, human); EC 2.7.1.37 (Akt1 protein, rat); EC 2.7.1.37 (Glycogen Synthase Kinase 3); EC 2.7.1.37 (Mitogen-Activated Protein Kinase 1); EC 2.7.1.37 (Mitogen-Activated Protein Kinase 3); EC 2.7.1.37 (Protein-Serine-Threonine Kinases); EC 2.7.1.37 (Proto-Oncogene Proteins c-akt); EC 2.7.1.37 (glycogen synthase kinase 3 beta)

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AN 2005254947 EMBASE

TI Cardiac overexpression of **melusin** protects from dilated cardiomyopathy due to long-standing pressure overload.

AU De Acetis M.; Notte A.; Accornero F.; Selvetella G.; Brancaccio M.; Vecchione C.; Sbroggio M.; Collino F.; Pacchioni B.; Lanfranchi G.; Aretini A.; Ferretti R.; Maffei A.; Altruda F.; Silengo L.; Tarone G.; Lembo G.

CS G. Tarone, Dept. of Genetics, Biology, and Biochemistry, Turin University, Via Santena, 5bis, 10126 Turin, Italy. guido.tarone@unito.it

SO Circulation Research, (27 May 2005) Vol. 96, No. 10, pp. 1087-1094. .
Refs: 27
ISSN: 0009-7330 CODEN: CIRUAL

CY United States

DT Journal; Article

FS 005 General Pathology and Pathological Anatomy
014 Radiology
018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry

LA English

SL English
ED Entered STN: 30 Jun 2005
Last Updated on STN: 30 Jun 2005
AB We have previously shown that genetic ablation of **melusin**, a muscle specific β 1 integrin interacting protein, accelerates left ventricle (LV) dilation and heart failure in response to pressure overload. Here we show that **melusin** expression was increased during compensated cardiac hypertrophy in mice subjected to 1 week pressure overload, but returned to basal levels in LV that have undergone dilation after 12 weeks of pressure overload. To better understand the role of **melusin** in cardiac remodeling, we overexpressed **melusin** in heart of **transgenic** mice. Echocardiography analysis indicated that **melusin** over-expression induced a mild cardiac hypertrophy in basal conditions (30% increase in interventricular septum thickness) with no obvious structural and functional alterations. After prolonged pressure overload (12 weeks), **melusin** overexpressing hearts underwent further hypertrophy retaining concentric LV remodeling and full contractile function, whereas wild-type LV showed pronounced chamber dilation with an impaired contractility. Analysis of signaling pathways indicated that **melusin** overexpression induced increased basal phosphorylation of GSK3β and ERK1/2. Moreover, AKT, GSK3β and ERK1/2 were hyper-phosphorylated on pressure overload in **melusin** overexpressing compared with wild-type mice. In addition, after 12 weeks of pressure overload LV of **melusin** overexpressing mice showed a very low level of cardiomyocyte apoptosis and stromal tissue deposition, as well as increased capillary density compared with wild-type. These results demonstrate that **melusin** overexpression allows prolonged concentric compensatory hypertrophy and protects against the transition toward cardiac dilation and failure in response to long-standing pressure overload. .COPYRGT. 2005 American Heart Association, Inc.
CT Medical Descriptors:
*congestive cardiomyopathy: DI, diagnosis
*heart left ventricle overload: DI, diagnosis
protein expression
protein determination
heart ventricle remodeling
transgenic mouse
echocardiography
heart ventricle septum
heart left ventricle contractility
wild type
signal transduction
enzyme phosphorylation
apoptosis
heart dilatation
heart failure
nonhuman
mouse
rat
animal experiment
animal model
controlled study
animal tissue
animal cell
article
priority journal
Drug Descriptors:
*binding protein: EC, endogenous compound
***melusin: EC, endogenous compound**
beta1 integrin: EC, endogenous compound
mitogen activated protein kinase 3: EC, endogenous compound
mitogen activated protein kinase 1: EC, endogenous compound
protein kinase B: EC, endogenous compound

glycogen synthase kinase 3alpha: EC, endogenous compound
unclassified drug
RN (mitogen activated protein kinase 3) 137632-07-6; (mitogen activated protein kinase 1) 137632-08-7; (protein kinase B) 148640-14-6

L3 ANSWER 4 OF 4 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2005:563501 SCISEARCH
GA The Genuine Article (R) Number: 930DR
TI Cardiac overexpression of **melusin** protects from dilated cardiomyopathy due to long-standing pressure overload
AU De Acetis M; Notte A; Accornero F; Selvetella G; Brancaccio M; Vecchione C; Sbroggio M; Collino F; Pacchioni B; Lanfranchi G; Aretini A; Ferretti R; Maffei A; Altruda F; Silengo L; Tarone G (Reprint); Lembo G
CS Univ Turin, Dept Genet Biol & Biochem, Via Santena, 5Bis, I-10126 Turin, Italy (Reprint); Univ Turin, Dept Genet Biol & Biochem, I-10126 Turin, Italy; IRCCS, Dept Angiocardioneurol, Pozzilli, IS, Italy; San Giovanni Battista Hosp, Expt Med Res Ctr, Turin, Italy; Univ Roma La Sapienza, Dept Expt Med & Pathol, Rome, Italy
guido.tarone@unito.it; lembo@neuromed.it
CYA Italy
SO CIRCULATION RESEARCH, (27 MAY 2005) Vol. 96, No. 10, pp. 1087-1094. ISSN: 0009-7330.
PB LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
DT Article; Journal
LA English
REC Reference Count: 27
ED Entered STN: 9 Jun 2005
Last Updated on STN: 9 Jun 2005
AB We have previously shown that genetic ablation of **melusin**, a muscle specific beta 1 integrin interacting protein, accelerates left ventricle (LV) dilation and heart failure in response to pressure overload. Here we show that **melusin** expression was increased during compensated cardiac hypertrophy in mice subjected to 1 week pressure overload, but returned to basal levels in LV that have undergone dilation after 12 weeks of pressure overload. To better understand the role of **melusin** in cardiac remodeling, we overexpressed **melusin** in heart of **transgenic** mice. Echocardiography analysis indicated that **melusin** over-expression induced a mild cardiac hypertrophy in basal conditions (30% increase in interventricular septum thickness) with no obvious structural and functional alterations. After prolonged pressure overload (12 weeks), **melusin** overexpressing hearts underwent further hypertrophy retaining concentric LV remodeling and full contractile function, whereas wild-type LV showed pronounced chamber dilation with an impaired contractility. Analysis of signaling pathways indicated that **melusin** overexpression induced increased basal phosphorylation of GSK3 beta and ERK1/2. Moreover, AKT, GSK3 beta and ERK1/2 were hyper-phosphorylated on pressure overload in **melusin** overexpressing compared with wild-type mice. In addition, after 12 weeks of pressure overload LV of **melusin** overexpressing mice showed a very low level of cardiomyocyte apoptosis and stromal tissue deposition, as well as increased capillary density compared with wild-type. These results demonstrate that **melusin** overexpression allows prolonged concentric compensatory hypertrophy and protects against the transition toward cardiac dilation and failure in response to long-standing pressure overload.
CC CARDIAC & CARDIOVASCULAR SYSTEMS; HEMATOLOGY; PERIPHERAL VASCULAR DISEASE
ST Author Keywords: **melusin**; cardiac hypertrophy; heart failure; signal transduction; fibrosis
STP KeyWords Plus (R): HYPERTROPHY IN-VIVO; **TRANSGENIC** MICE; GENE; DYSFUNCTION; EXPRESSION; FAILURE; MECHANISMS; INHIBIT; RAT
RE

Referenced Author	Year	VOL	ARN PG	Referenced Work

(RAU)	(RPY)	(RVL)	(RPG)	(RWK)
ANTOS C L	2002	99	907	P NATL ACAD SCI USA
BADORFF C	2002	109	373	J CLIN INVEST
BONCI D	2003	10	630	GENE THER
BRANCACCIO M	1999	274	29282	J BIOL CHEM
BRANCACCIO M	2003	9	68	NAT MED
BRODAL P	1977	232	705	AM J PHYSIOL
BUENO O F	2002	91	776	CIRC RES
BUENO O F	2000	19	6341	EMBO J
CONDORELLI G	1999	99	3071	CIRCULATION
CONDORELLI G	2002	99	12333	P NATL ACAD SCI USA
DATTA S R	1999	13	2905	GENE DEV
DIFFEE G M	2003	284	H830	AM J PHYSIOL-HEART C
ESPOSITO G	2002	105	85	CIRCULATION
FREY N	2003	65	45	ANNU REV PHYSIOL
GALLAGHER A M	1998	32	84	HYPERTENSION
GELPI R J	1991	68	555	CIRC RES
GULICK J	1991	266	9180	J BIOL CHEM
HAASE D	2002	4	23	EUR J HEART FAIL
HUNTER J J	1999	341	1276	NEW ENGL J MED
JUHASZOVA M	2004	113	1535	J CLIN INVEST
KADDOURA S	1996	93	2068	CIRCULATION
LEW A M	1999	341	647	BIOCHEM J 3
LIPS D J	2004	109	1938	CIRCULATION
TSCHOPE C	2004	18	828	FASEB J
VECCHIONE C	2002	105	1700	CIRCULATION
WOLSKA B M	1996	271	H1250	AM J PHYSIOL-HEART C
ZILE M R	2002	105	1503	CIRCULATION

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     FILE 'BIOSIS, MEDLINE, EMBASE, SCISEARCH' ENTERED AT 10:29:37 ON 25 APR
     2006
L1            75 S MELUSIN
L2             0 S L1 AND TRASNGENIC
L3             4 S L1 AND TRANSGENIC

=> s l1 and transgen
=>
=> d his

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L1            75 S MELUSIN
L2             0 S L1 AND TRASNGENIC
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